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Systemic Inflammatory Response in Patients with Acetic Acid Poisoning

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Abstract: The authors investigated the interdependence of fibrinolysis activation and systemic inflammatory response syndrome development in 68 patients with severe level of acute poisoning by azaleptin. A positive correlation was found between blood levels of D-dimer, clinical indications of systemic inflammation and C-reactive protein.

Keywords: acute poisoning, systemic inflammatory response syndrome, fibrinolysis, D-dimer.

Acetic acid (AC) differs from other cauterizing substances not only by the nature of the burn, but also by its active resorptive properties, leading to the development of hemolysis, toxic coagulopathy, multiple intravascular coagulation syndrome and critical condition of the body. Hemolysis of erythrocytes is one of the leading triggers in the development of toxic coagulopathy syndrome. Burn destruction of tissues, disintegration of erythrocytes cause the release of a large amount of thromboplastic material and the beginning of stage I of toxic coagulopathy - the stage of hypercoagulation [2]. Early detection of signs of disseminated intravascular coagulation (DIC) [1,2], along with an assessment of the development of inflammation, will prevent the development of complications, increase the effectiveness of their prevention and, ultimately, determine the outcome of poisoning.

The aim of the study was to identify the relationship between the activation of fibrinolysis and the development of systemic inflammatory response syndrome in patients with acute acetic acid poisoning.

Materials and methods of research. 68 patients (average age 34.6+4.4 years) with acute acetic acid poisoning were examined. admitted to the intensive care unit of the Bukhara branch of the RNCEMP. The comparison group consisted of 30 healthy volunteers (average age 30.1±7.9 years). The level of consciousness was determined by the Glasgow scale. Violation of consciousness to the level of coma of the III art. with poisoning of the criminal code was accompanied by the development of acute respiratory failure of mixed genesis. 12 people died in the somatogenic phase of poisoning as a result of hypoxic brain damage with increasing phenomena of multiple organ failure (PON) and severe sepsis.

Depending on the outcome of the disease, all patients were divided into two groups: group 1 - survivors (n=56); group 2 — deceased (n=12).

The development of systemic inflammatory response syndrome (SERS), sepsis was determined by the presence of signs presented in the recommendations of the International Guidelines on Severe Sepsis and Septic Shock (The Third International Consensus Definitions for Sepsis and Septic Shock - Sepsis-3 (2016)). The severity of multiple organ failure (PON) associated with sepsis was assessed according to the SOFA scale (1994) adopted by the European Society of Intensive Care. The content of C-reactive protein (SRB) in blood serum was also studied by the latex method of Bio Systems (Spain). Fibrin degradation products (PDF) were determined by an immunological method based on agglutination of latex particles coated with monoclonal antibodies on a biochemical analyzer STA - Compact coagulometer by Roche (Switzerland) using COBAS INTEGRA Tinaquant D-Dimer (D-DI) kits. For early prediction of the outcome of critical conditions caused by acute poisoning, the coagulographic death index (KILI) was calculated based on the indicators of the electrocoagulogram of arterial and venous blood on the

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day of admission of the patient to the hospital according to the formula:

KILI=T 2a - T 2b - 500 (T 1a/T 2a) + 270,

where T 2a and T 2b are the time of clot formation in arterial and venous blood (c), T 1a is the time of clotting activation in arterial blood (c).

Negative values of the KILI indicated a high probability of death (more than 90%). KEEL values in the range from 0 to +100 corresponded to the danger zone with a probability of death of about 50%. Values exceeding +100 corresponded to a low probability of death (less than 15%).

Results and their discussion. The development of CVD and an increase in the number of SOFA scores were observed in all victims with acute poisoning of the criminal code (two or more signs corresponding to the Bone classification) (1992) [6]. In the group of deceased patients, in contrast to the survivors, there was an increase in signs of multiple organ failure (an increase in the SOFA index on the 3rd and 5th days) and more pronounced manifestations of CVD. The increase in organ dysfunction in patients of this group was mainly associated with the suppression of the functions of the central nervous system and respiratory organs.

Thus, the severity of the condition in the two groups of patients examined by their homogeneity was determined by the depth of the comatose state, which caused the development of mixed hypoxia, disorders in the blood clotting system, the development of systemic inflammation. Both groups had their own clinical features: in group 1, 46% of patients had foci of purulent infection in the lungs (focal, lower lobe pneumonia) without signs of severe sepsis, in group 2 (deceased), in 100% of cases, purulent infection resulted in bilateral drain pneumonia, signs of severe sepsis with the development of systemic inflammation and more pronounced disorders in the blood clotting system were observed.

Among the potentially possible markers of activation of blood clotting and the development of disseminated intravascular coagulation (DIC), the greatest attention is paid to the study of those that reflect the formation and cleavage of fibrin - products of fibrin degradation (D-dimer) [1]. The high specificity of the D-dimer is due to the fact that its presence indicates the formation of thrombin in an amount that provides not only the cleavage of fibrinopeptides, but also the activation of factor XIII (fibrinase) [2]. In deceased patients who had a coagulographic index of 204.4 ±56.8 units at admission, an increase in the level of D-dimer in blood plasma was observed by 12.4 times compared to the norm already 1 day after poisoning and its further increase throughout the entire observation period. In the surviving patients, the increase in the D-dimer content in the blood was less pronounced and was observed from 1 to 3 days after admission. A comparative assessment of the D-dimer in the blood of patients of the 1st and 2nd groups revealed significant differences on the 1st, 3rd and 5th days of follow-up. A negative correlation of the D-dimer content in blood plasma with the KILI value from day 1 to day 5 was also revealed (day 1 g = -0.65; day 3 g= -0.78; day 5 g= -0.91, n=27, $p<0.05$). Thus, in all patients with acute poisoning of the criminal code, laboratory signs of the development of DIC syndrome were observed, more pronounced in patients with a fatal outcome.

The presented data on the number of signs of CVD in patients with acute poisoning indicated more pronounced inflammation in deceased patients. A study of the C-reactive protein content in the blood also confirmed a higher level of inflammation activity in deceased patients. At the same time, the content of D-dimer in the blood plasma of patients significantly correlated with the number of signs of CVD on the 1st and 5th days (g=0.47, $p<0.05$ and g=0.79, $p<0.05$, p=27), the content of SRV in the blood serum (g=0.51, $p<0.05$, n=26) and the number of SOFA points on the 5th day after poisoning (g=0.86, $p<0.05$, n =26). As shown earlier [2], mixed-type hypoxia developing in patients with acute acetic acid poisoning, in turn, provokes the development of bacterial infection, both local (group 1) and systemic (group 2). Bacteremia, confirmed by a bacteriological blood test, was observed in 35% of patients with a negative prognosis of the outcome of the disease. The presence of pathogenic microflora in the victims, which includes microflora detected in biological media (Staphylococcus aureus (55%),

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Pseudomonas aeruginosa (45%), proteus, coagulase-negative staphylococcus), indicates pronounced violations of humoral immunity, immunity of the mucous membranes. Inflammation caused by induced hypoxia of mixed type in combination with foci of infection, disorders in the blood clotting system, together depending on the depth and severity of the disturbance of consciousness, cause an increase in manifestations of multiple organ failure (dysfunction).

In group 1 patients, standard detoxification and immunotropic therapy led to an improvement in the condition against the background of a decrease in the level of inflammation, whereas in group 2 patients, the development of systemic inflammation with persistent disorders in the blood clotting system and progressively increasing organ dysfunction was observed, which ultimately led to death.

Thus, patients resistant to standard therapy for acute poisoning of the criminal code need correction of the blood clotting system and the activity of inflammation in the early period, which consists in a new approach to treatment with the additional use of synthetic fibrinolysis inhibitors, anticoagulants in combination with immunotropic therapy.

Conclusions

1. An increase in the level of D-dimer in blood plasma is observed in all patients with acute acetic acid poisoning, which indicates the activation of coagulation and fibrinolysis processes in this category of patients.

2. In patients with acute poisoning with acetic acid, a positive correlation of the -D-dimer in blood plasma with the number of clinical signs of CVD and the level of C-reactive protein was revealed.

3. In the group of deceased patients, the content of D-dimer and C-reactive protein remained significantly higher and signs of organ dysfunction increased

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